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Prickle Cell Hyperplasia in the Snout of the Redhorse Sucker
(*Moxostoma aureolum*) Associated with an Infection by the
Myxosporidian *Myxobolus moxostomi* sp. nov.

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(Plates I-III).

INTRODUCTION.

The corium is the primary site of infection for many histozoic myxosporidians. Tissue responses to such infection are usually manifest as inflammatory reactions and hyperplasia of connective tissue which often form the cysts enclosing the developing spores (Kudo, 1919, 1929, 1930, 1934; Nigrelli and Smith, 1938). The degree of these responses varies with the host, parasite and site of infection. In certain instances the connective tissue cells become highly modified, transforming into epithelioid-like cells. Such a case was reported by Kudo (1929, 1934, 1946) for the connective tissue cells in the corium of the minnow, *Pimephales* = *Hyborhynchus*) *natatus*, infected with *Thelohanellus* (*Myxobolus*) *notatus* (Mavor). Occasionally a hyperplasia of the epithelium is also associated with these infections (Nigrelli and Smith, 1940), although in most cases there is a tendency for this structure to become thin and sloughed as a result of pressure induced by the enlarging underlying cysts.

The present contribution deals with a new species of myxosporidian, *Myxobolus moxostomi*, found in the corium of the snout of the northern redhorse sucker, *Moxostoma aureolum* (LeSueur), collected in Illinois. Associated with this growth is an hypertrophy of the overlying epithelium in which prickle cells are the main cellular elements involved.

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DESCRIPTION OF THE PARASITE.

The affected fish showed a marked swelling in the midregion of the snout just above the maxilla. No external cysts were visible. The fish was sacrificed and the swollen part excised and fixed in 10% neutral formalin. Paraffin sections were cut at six microns and stained with hematoxylin-eosin, Mallory's, Masson's and Giemsa's methods.

The infection was localized in the corium. The cysts were composed of delicately developed connective tissue, and were more or less confluent (Plate I, figure 1). Those that were discrete measured from 0.5 mm. to

4 mm. The central part of the cysts contained numerous spores, a smaller number of developing disporoblastic pansporoblasts and cellular debris.

Like the other members of the genus *Myxobolus*, the parasites are polysporous. The spores (Plate I, figure 2) varied in shape from circular to ovoid in front view and were fusiform, with more or less rounded tips in side view. They measured $6.2-9.4 \times 5.5-9.4 \times 3.1-4.7$ microns (average, $7.6 \times 7.2 \times 3.9$ microns). It can be assumed that the living spores were slightly larger than the measurements indicated since some shrinkage must have occurred during fixation. The shell was of uniform thickness, with several darkly-staining bodies present in many of the (immature?) spores. The sutural ridge and line were not especially conspicuous. The polar capsules were pyriform in shape, often convergent. They measured $2.3-3.9 \times 1.6-3.2$ microns (average, 3.6×2.3). The polar filament, when evident, showed from three to five spiral turns. The sporoplasm was either uninucleate or binucleate. An iodophilous vacuole was evident in many spores.

This parasite was tentatively identified by Nigrelli (1943) as *Myxobolus conspicuus* Kudo. However, a more detailed comparison of the present species with those reported by Kudo (1934) from the same and related fishes is given in Table I. It differs from them in shape and size of spore, details in structure of shell, site of infection and type of tissue responses elicited. The most striking difference is found in measurements of the various parts of the spore. It is for these reasons that it is considered a new species for which the name *Myxobolus moxostomi* is given.

PATHOLOGY.

The actual site of infection with *Myxobolus moxostomi* was in the deeper layers of the corium. Host response in this region was shown by the development of a delicate fibrous growth around the trophozoite and spore masses. There was some invasion and destruction of muscle and periosteal tissues, accompanied by a mild inflammatory reaction. Corial melanophores were at a minimum.

TABLE I.
Comparison of Species of *Myxobolus* from *Moxostoma*.

PARASITE:	<i>M. moxostomi</i> sp. nov.	<i>M. congesti-</i> <i>cus</i> Kudo, 1934	<i>M. vastus</i> Kudo, 1934	<i>M. gravidus</i> Kudo, 1934	<i>M. conspicuus</i> Kudo, 1929
HOST:	<i>M. aureolum</i>	<i>M. anisurum</i>	<i>M. aureolum</i>	<i>M. anisurum</i>	<i>M. breviceps</i>
SITE OF INFECTION:	Corium of head Deep	Fins Superficial	Corium, body scales Superficial	Skin, fins Superficial	Skin of head Superficial
TUMOR:	Present	Present	Present
SIZE OF CYSTS:	0.5-3 mm.	0.3-1 mm.	2.5-3.8 mm.	0.5 mm.	0.5-4 mm.
CONNECTIVE TISSUE DEVELOPMENT:	Minimum	Minimum	Hypertrophy	Hypertrophy
EPIDERMIS:	Hypertrophy	Thin, broken	Thin	Thin, unicel- lular glands absent
VASCULARIZATION:	Poor (region around tro- phozoites)	Rich	Rich
SPORE SHAPE FRONT:	Circular to ovoid	Circular to oval	Oblong	Oval to oblong	Oval
SIDE:	Fusiform, rounded ends	Lenticular	Lenticular	Fusiform	Fusiform
SPORE SIZE:	6.2-9.4 × 5.5- 9.4 × 3.12- 4.68 micra	9-10 × 8.5- 9.5 × 6 micra	9.5-10.5 × 7.5- 8 × 4-4.5 micra	12-14 × 9.5- 10 × 7 micra	9-11.5 × 6.5- 8 × 4.5-5.5 micra
SHELL:	Mod. thick	Mod. thick, radiating folds	Mod. thick 7 radiating lines	Mod. thick, 4-6 folds in post. part	Mod. thick No striations
SUTURAL RIDGE:	Not promi- nent	Wide	Thickened at ends	Inconspic- uous	Wider than thickness of shell
SUTURAL LINE:	Present	Indistinct	Indistinct	Straight
CAPSULE SHAPE:	Pyriform, slightly divergent	Pyriform, convergent	Pyriform	Pyriform, convergent	Pyriform, dissimilar in size
SIZE:	2.3-3.9 × 1.56- 3.2 micra	5-6 × 2.5-3.5 micra	4.5-5.5 × 1.5 -2.5 micra	5-5.5 × 2.5 micra	5-7 × 2-2.5 micra
FILAMENT:	3 to 5 turns	10 turns

The most striking changes, however, were noted in regions immediately above and adjacent to the actual site of infection (Plate II, figure 3). The fibrous growth here was considerably thickened and more vascularized. In some areas of the growth, the connective tissue appeared to give rise to a supporting stroma of the hypertrophied stratified epithelium. The cellular elements of the latter were mainly prickle cells showing prominent intercellular bridges (Plate III, figure 5). In some parts of the growth the cells appeared as normal polygonal-shaped structures with finely granular cytoplasm and nucleoplasm; the latter occasionally contained several deeply stained, peripherally placed granules of various sizes. The majority of nuclei also possessed a single

nucleolus and few cells showed two such bodies. In most cells the staining reaction of the nucleus was not intense with hematoxylin, but isolated groups of prickle cells were encountered in which this structure was definitely hyperchromatic. Although no mitotic figures were found there was some evidence of activity. In certain regions the cells had broken through the basement membrane and invaded the corium (Plate II, figure 4). The invasion, however, was not extensive.

In other regions of the growth there was considerable evidence of degeneration. The prickle cell growth had crowded and destroyed the mucous gland cells of the skin. The outermost layer of prickle cells showed the greatest changes, manifested metachromatically

with Masson's and Giemsa's stains. The nucleoli of these cells were larger and often led the entire nucleus. These regions also showed considerable infiltration by macrophages.

DISCUSSION.

Kudo (1929) and Nigrelli and Smith (1940) have shown that histozoic myxosporidian parasites may induce a thickening of the epithelium in the regions of the infection. Auerbach (1909) also found that the histozoic parasite *Myxidium bergense* may cause a thickening of the epithelium of the gall bladder of *Gadus virens*. In these cases the parasites were in more intimate contact with the tissues involved. In the redhorse sucker, the epithelial hyperplasia was immediately above but some distance away from the infection. Whether or not there was any direct association between the infection and the hyperplastic growth is difficult to say. Intercellular and intracellular cnidosporidians elaborate proteolytic enzymes and other chemical substances which may be responsible for considerable cellular degeneration, cell hypertrophy and other tissue responses noted in these infections. It is together possible that chemical substances elaborated by the developing parasites may have a stimulating growth factor which may diffuse to more distant areas either directly or through the circulation.

SUMMARY.

A new species of myxosporidian, *Myxobolus moxostomi*, from the corium of the mouth of the common redhorse sucker, *Moxostoma aureolum* (LeSueur), is described.

Hyperplasia of the connective tissue and stratified epithelium is associated with the infection. In the hyperplastic epithelium, prickle cells are the main cellular elements involved.

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EXPLANATION OF THE PLATES.

PLATE I.

- Fig. 1. Section through cysts showing numerous spores of *Myxobolus moxostomi* together with a few pansporoblasts and other cellular elements. Giemsa. $\times 75$.
- Fig. 2. Mature and immature spores of *Myxobolus moxostomi*. Giemsa. $\times 675$.

PLATE II.

- Fig. 3. Section through the overlying growth showing extensive development of corial fibrous tissue and hyperplastic epithelium. Hematoxylin-eosin. $\times 75$.
- Fig. 4. Basal columnar cells of the hyperplastic epithelium showing infiltration into the corium. Hematoxylin-eosin. $\times 675$.

PLATE III.

- Fig. 5. Prickle cells of the hyperplastic epithelium. Hematoxylin-eosin. $\times 675$.

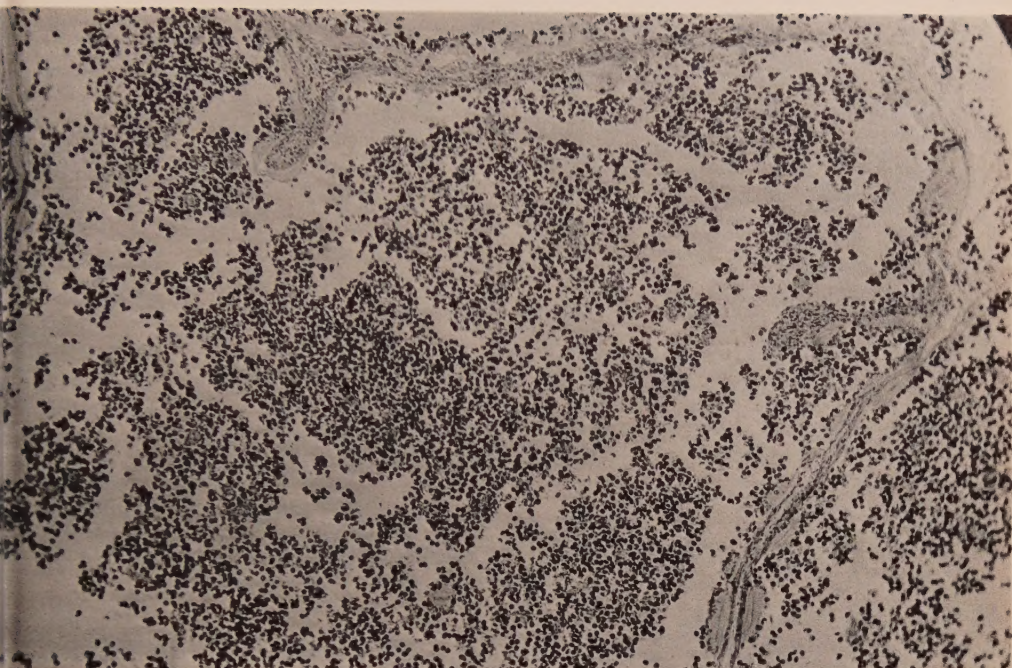


FIG. 1.

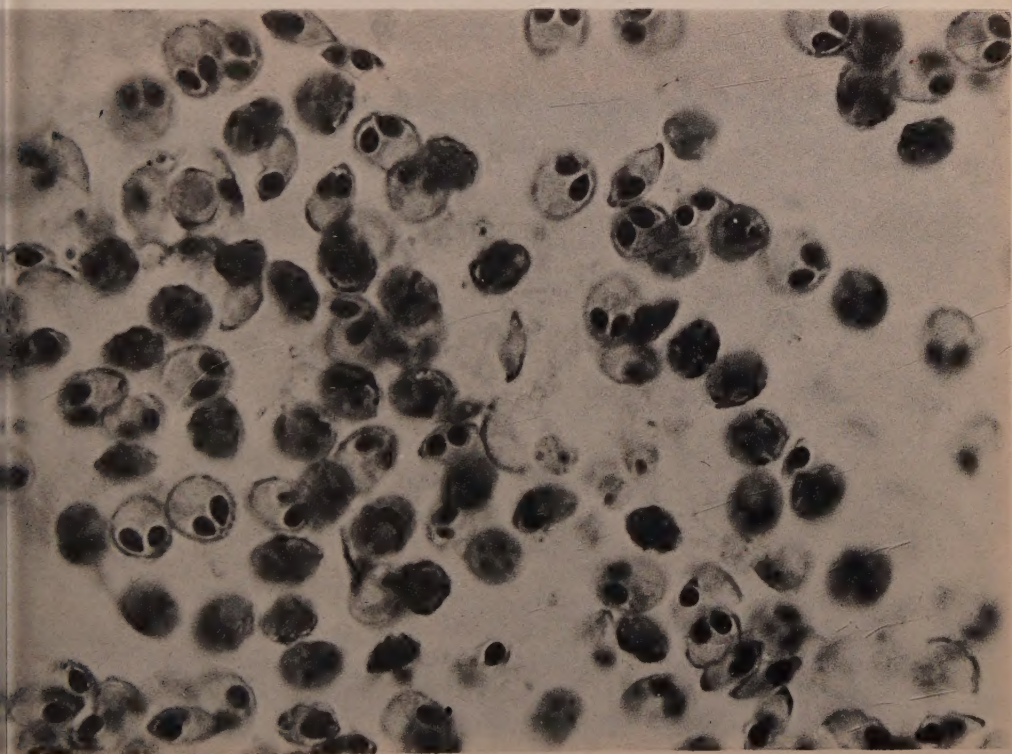
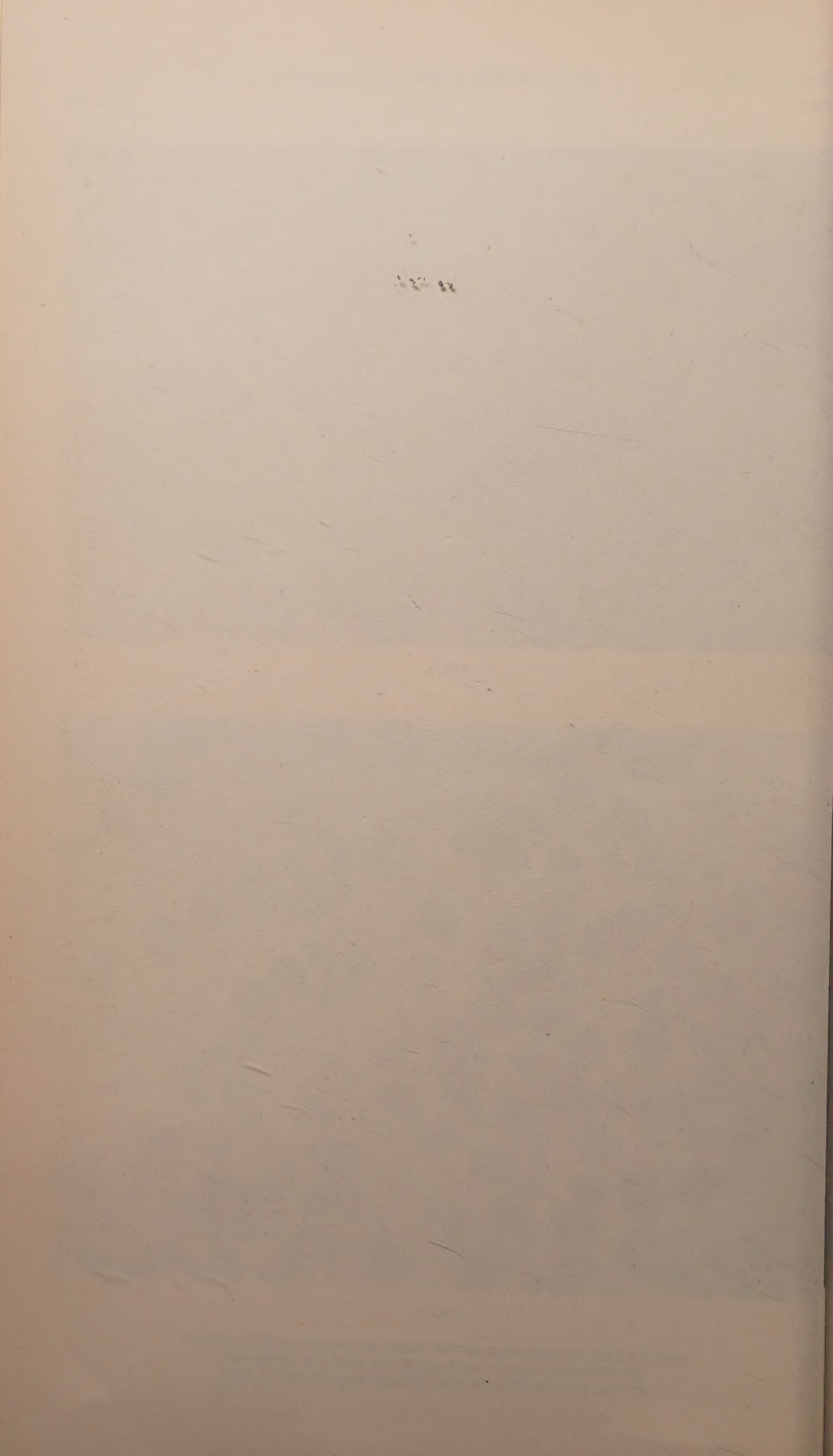


FIG. 2.

PRICKLE CELL HYPERPLASIA IN THE SNOUT OF THE REDHORSE SUCKER
(*MOXOSTOMA AUREOLUM*) ASSOCIATED WITH AN INFECTION
BY THE MYXOSPORIDIAN, *MYXOBOLUS MOXOSTOMI* SP. NOV.



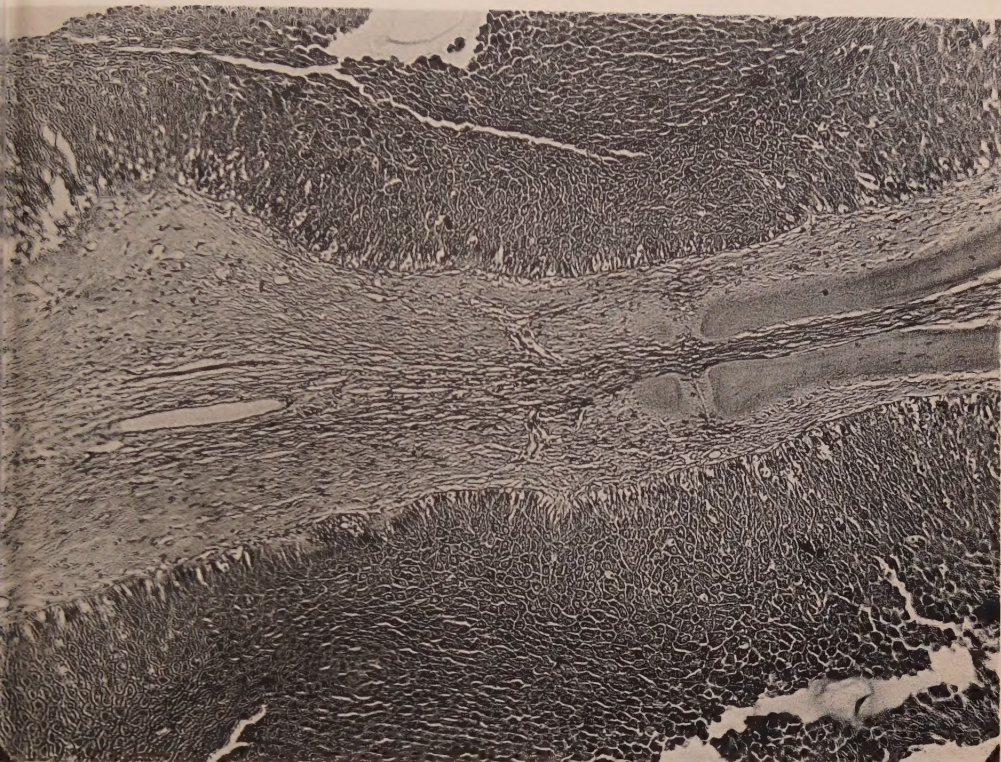


FIG. 3.



FIG. 4.

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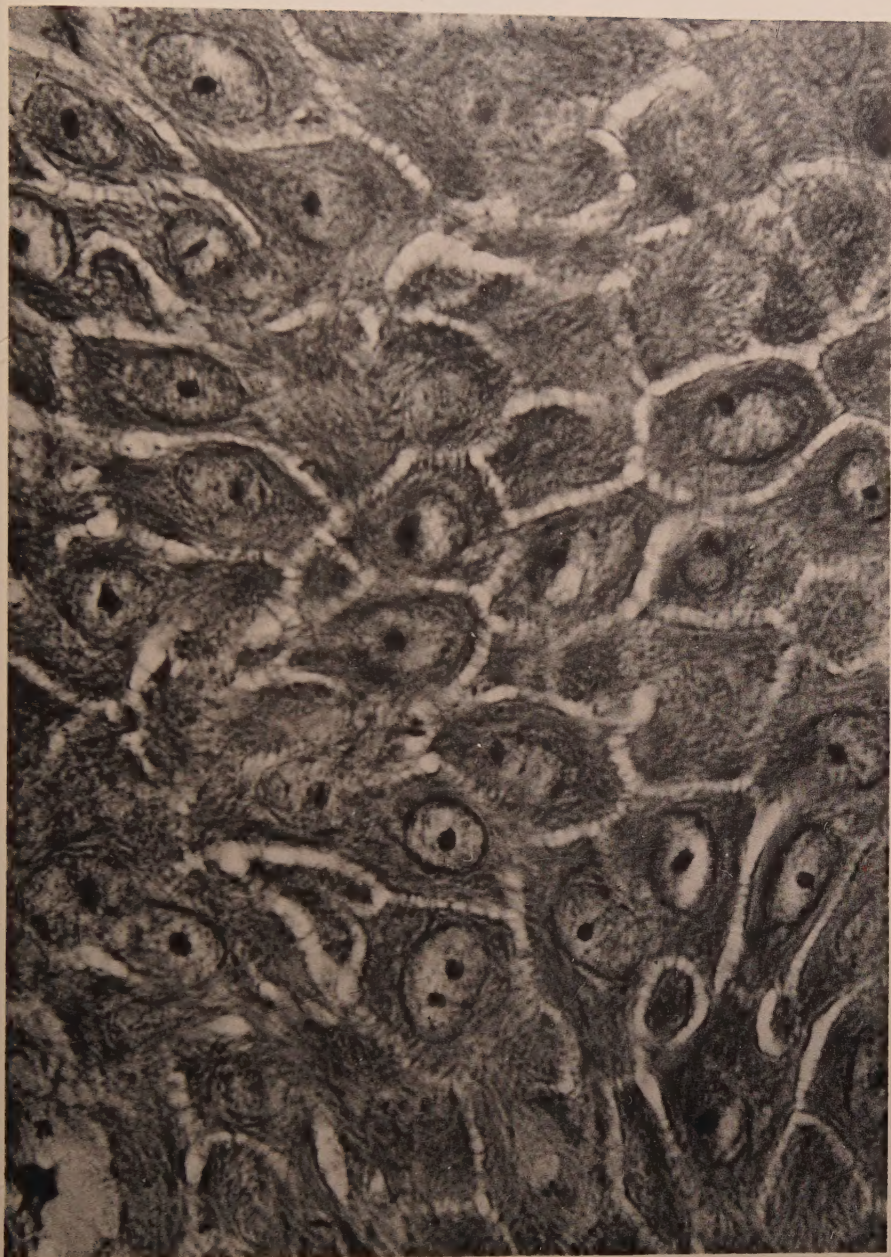


FIG. 5.

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